Volume 29. November, 1902. No. 11.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

PARADOXICAL PSEUDO-HYPERTROPHY FOLLOWING INFANTILE CEREBRAL HEMIPLEGIA.

By L. Pierce Clark, M.D.
NEW YORK.

The hypertrophy of paralyzed parts in infantile cerebral palsy is a rare condition. No special article on the subject exists in literature, and no cases illustrative have been reported in America, although several observers, such as Sachs, Peterson and Osler have made diligent search for them. The different degrees of muscular and bone atrophy in spinal palsy of children have been well studied and satisfactorily explained on a basis of a disturbance in the trophic center of the anterior horns of the cord, the site of the destructive palsy lesion. Notwithstanding the almost uniform presence of atrophy in spinal palsies (acute anterior poliomyelitis) Sellegmuller has described a bony elongation in spinal palsies, and Dejerine claims to have demonstrated in such cases true fiber hypertrophy in those muscles totally paralyzed. Joffroy and Achard have also proven the truth of Dejerine's position in spinal palsy cases, even 40 or 50 years after the original onset of the lesion. However well the positive exclusion of a few nerve cells from the destructive lesion in the cord might satisfy the hypothesis that by their overaction fiber hypertrophy might occur, no such explanation could be urged for the hypertrophic condition found in cerebral palsy cases, as there are no trophic centers in the brain as such. If such functions are exercised by the brain they must be through muscular movements represented in the cortex. A normal musculature in cerebral palsy cases is common and easily explained on the theory of a slight or transitory brain involvement and intact anterior horn cells of the cord, but the case assumes perplexing aspects in the extreme when actual hypertrophy of paralyzed parts occurs after the initial paralytic stroke.

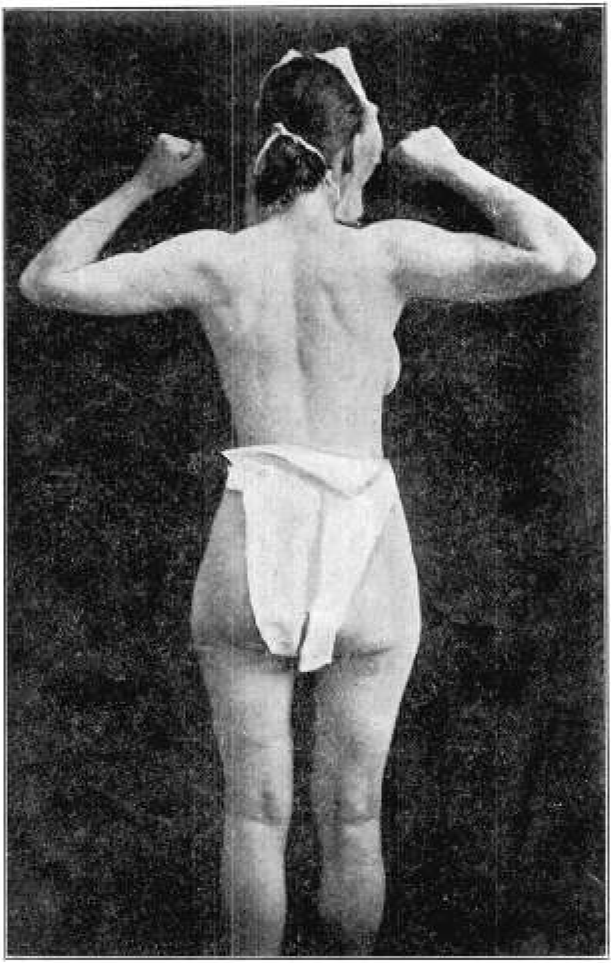
Brief Historical Review—The voluminous literature of infantile cerebral palsy deals largely with the condition while the patients are still in infancy, and as hypertrophy probably requires some years for its development we cannot expect to note many recorded cases. Moreover, but 30% to 50% of essential palsies develop epilepsy, therefore less than one-half of adult cases come under careful observation, and then too for epileptiform attacks usually. The existence of the latter symptoms often fails to excite the clinician's attention to the initial organic mischief, as hopes of ameliorating a lesion so long existent are not often entertained. Then, too, most cases of hypertrophy are found in literature on athetosis, notwithstanding in the twelve recorded cases (five my own) but eight had athetosis.

In the pioneer study of hypertrophy in athetosis, Hammond in 1871 describes no case dating from infantile cerebral palsy or from childhood even. Clay and Shaw also failed to find hypertrophy in his careful study of choretic paresis in 1873. but Bernhardt has the honor of describing the first case in 1876, the same year in which Gowers also reports a case, or possibly two, although the occurrence of the second is doubtful. In 1878 Oulmont, in a thesis on athetosis, includes one case of hypertrophy, and Goudard described one case in 1884 in a general article upon cerebral palsies in children. In an extensive article on athetosis, Berger in 1885 stated that a small percentage of adult athetosis show the state of hypertrophy, which he believed to be an inexplicable freak of pathology. In 1891 Schieber reported an interesting and minutely-described case, and Kaiser, in 1897, recorded a unique case of marked hypertrophy of muscles and bone, complicated by myotonia. The chronological order of the cases of hypertrophy on record, including three reported for the first time in this paper, are as follows: 1876, Bernhardt, 2, athetosis present; 1876, Gowers, 1, athetosis present; 1878, Oulmont, 1, athetosis present; 1884, Goudard, 1, athetosis present; 1891, Schieber, 1, athetosis present; 1897, Kaiser, 1, athetosis present; 1900, Clark, 2, athetosis and epilepsy present in one; epilepsy without athetosis present in the other; 1902, Clark, 3, epilepsy present in all without athetosis.

The family history of the hypertrophic form of infantile cerebral palsy presents no marked neuropathy, although in cases reported by Bernhardt, Schieber, Kaiser and myself, convulsive disorders such as epilepsy, tic disease and chorea were found in the collateral family stock.

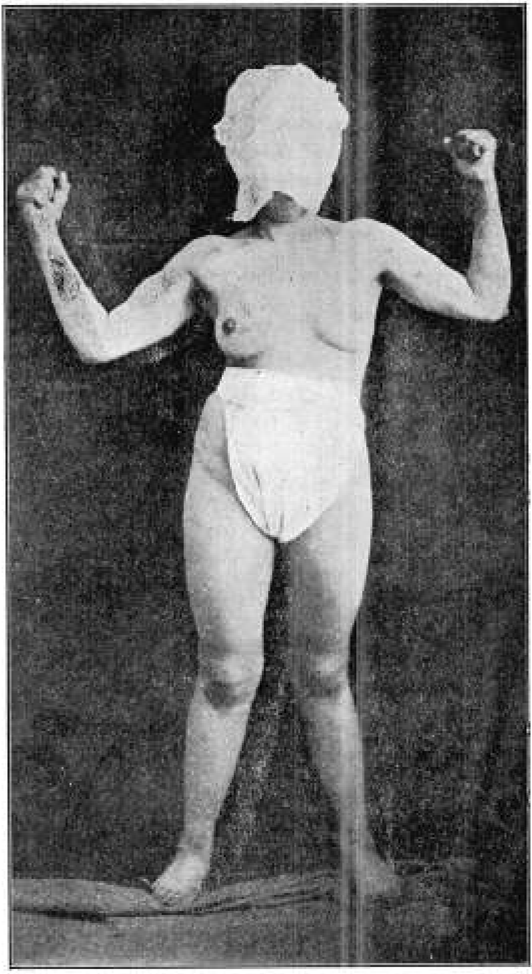
The intra-uterine life of the patient has proven uneventful in all cases except some slight and insignificant emotional disturbance in the mother. Labor has always been at term and no evidence of trauma or severe asphyxiation has been found to exist. Instrumental delivery was practiced in two cases, but no injury resulted. All the children were heathful until the time of the stroke, except in two who developed infantile eclampsia at dentition. The cases of hypertrophy are equally divided between the two sexes. The age of onset of the paralytic stroke in cases in which hypertrophy developed later ranges as follows: two at 18 months; three at four years; three at six years; three at seven years; one at fourteen years; an average of six years of age, considerably later than in ordinary palsy cases (under 3 1-2). The causes ascribed for the palsy in the 12 cases have been: four, scarlet fever, or its sequela; two, errors of dentition; and in six none, as in ordinary palsy cases. The immediate excitant to the stroke in all cases was too trivial for consideration.

Usually the more important symptoms of the onset of cerebral palsy have been absent in the hypertrophic cases, but in all of mine there were more or less severe convulsions, vomiting, fever and physical prostration. In two cases reported by Bernhardt and Kaiser the palsy occurred without apparent knowledge of it on the part of the patient. The presence of palsy was manifest on awakening in the morning. Usually there was considerable prostration following the stroke and the patients were bed-ridden for several days, weeks or months. The preferment of the hemiplegia for the right or left side is relatively the same as that for ordinary



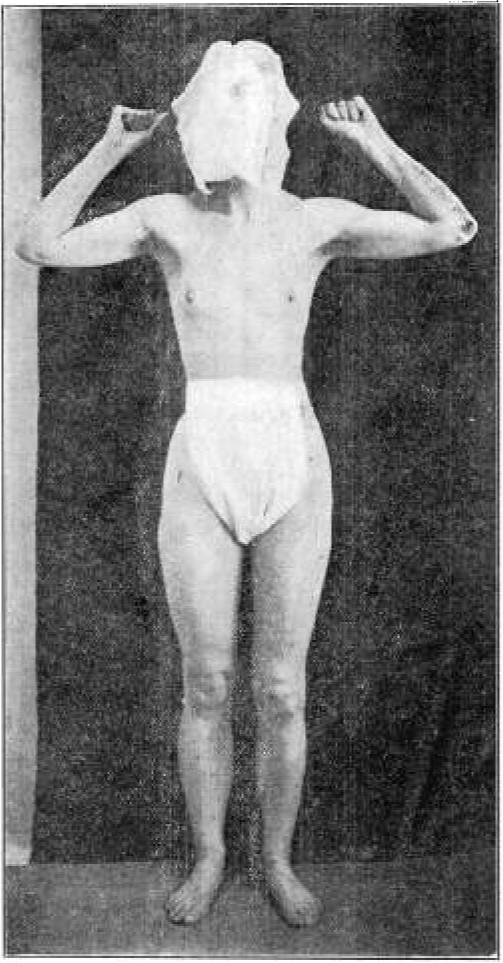
Case I. E. P., paradoxical pseudo-hypertrophy in infantile cerebral palsy. Relative enlargement of right shoulder, arm and thigh. Right arm (paralyzed) at biceps 3-4 inches larger than left or well side. Left handed. Epilepsy (partial) and athetosis.

palsy (7 to 5) in favor of the right side. The extent of paralysis has always been hemiplegic. The leg and face recover most rapidly and completely, as is usual in ordinary palsy cases (a few days to several weeks). Athetosis was present in eight cases; its onset occurred on or before the patient re-



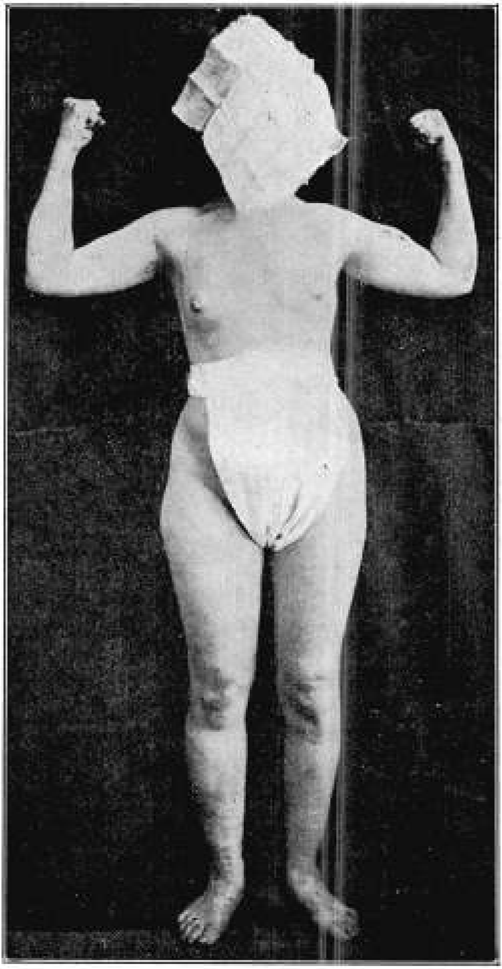
Case II. F. W., paradoxical pseudo-hypertrophy in infantile cerebral palsy. Relative enlargement of entire right side, including face. Hypertrophy most marked in right arm 3-4 inch. Deltoid and biceps most hypertrophic. Epilepsy (partial) but no athetosis. Left handed.

covered from the prostration of the stroke (choreic paresis) but in the two cases reported by Schieber and Gowers, athetosis occurred in the one five weeks after the cerebral lesion, while in the other it developed one year after the stroke (post-



Case III. J. P., paradoxical pseudo-hypertrophy in infantile cerebral palsy. Relative enlargement of left shoulder and arm (paralyzed); arm and left shoulder 1-2 in. larger. Muscles hypertrophic; deltoid, biceps and triceps. Forearm and lower extremity of the same side markedly atrophic (1-2 to 1 in.). Epilepsy (partial) but no athetosis; right handed.

hemiplegic athetosis). The character of the spontaneous movements are generally described as typical of Hammond's athetosis (post-hemiplegic athetosis or chorea), but frequently it resembles choreic paresis (fibrillary chorea); the associ-



Case V. A. V., paradoxical pseudo hypertrophy in infantile cerebral palsy. Relative enlargement of left arm (hemiplegic side) 1-4 in.; hypertrophy confined mostly to biceps. Atrophy of forearm and lower extremity of left (paralyzed) side (1-2 to 1 1-2 in.). Epilepsy (partial) but no athetosis; hight handed.

ation of the two forms are often seen in the same case. The movements are most marked in the forearm and shoulder muscles. In about one-half the cases the lower extremity engages in the athetosis in a minor degree, although in Bernhardt's second case the leg alone was the seat of athetosis and hypertrophy. The athetosis ceases usually in a position of rest or while asleep. Voluntary effort at control may decrease the movements, rarely it increases; voluntary acts bring on increased athetotic movements. The posture of the arm is usually that of partial rest with hand pressed against the side and elbow slightly extended, although the posture and movements, as in essential athetosis, may vary greatly.

The mental state of the patients on the whole is better than usually obtains in ordinary palsy cases. While the intellectual element as such is quite lacking, the patients exhibit considerable ability in learning industrial pursuits. Those cases in which athetosis exists and those in which it does not occur are about equal in ability to undertake trades. Possibly the non-athetotic are slightly duller students, but this defect offsets the physical agitation of the affected parts in the athetotic class.

The length of time necessary to produce the hypertrophy is obviously difficult to ascertain, but the time elapsing between the palsy lesion and the detection of the hypertrophy has been very variable, from 8 to 29 years, the average being 16 years. The age of the patients at the time the hypertrophy has been recognized ranges from 12 to 36 years, the average being 22 years. The hypertrophy may involve any and all parts that are paralyzed, even the breast and testicles may enlarge. The skin, muscles and fat may all hypertrophy in a single case, although some degree of muscle hypertrophy is uniformly present in every case. The enlargement has had its seat in the upper extremity in every instance except in Bernhardt's second case, where the leg was hypertrophic. The arm has shown most enlargement in nine cases, the forearm in two and the calf in one. The muscles most frequently hypertrophied are found in the following order of occurrence: biceps, deltoid and triceps.

The parts not hypertrophic follow the general law for atrophies in ordinary cerebral palsy cases. The hypertrophied tissues have not been studied carefully from the histological standpoint in any cases except in two of our own; in these true fiber hypertrophy existed. An enlargement and increase in the number of the muscle spindles was found; usually hypertrophy and hyperplasia went hand in hand. No doubt many of the reported cases of enlargement of an extremity should be classed as fat hypertrophy, as noted in a case by Gibatteau, a fact also known to Cazauvielh. In such cases the excessive fat appears to be compensatory for the muscular atrophy, as has been shown in poliomyelitis (Freud). Our suspicions are further strengthened inasmuch as the hypertrophic extremity is without exception relatively weaker than its opposite on the healthy side (pseudo-muscular hypertrophy). Too much stress, however, should not be laid on this fact, as the contractile power of any muscle in different individuals is not always in definite ratio to its volume. The difficulties of examining excised muscular tissue for fiber hypertrophy, either from the living or dead subject, have been well emphasized by Batten, Spencer and Gowers. In most cases the skin and fatty tissue have shown hyperplasia of their essential elements[1].

Bony hypertrophy was first actually proven by me in one of my cases (1899) by X-ray examination, and still more recently Lannois and Fayolle have reported the same results by the same method. The bones of the entire upper extremity may be hypertrophic, although the bones usually found enlarged are those corresponding to the hypertrophic soft parts which they underlie. The degree of circumferential enlargement of the hypertrophic extremity varies from 1-4 to 7-8 in., although not usually more than 1-2 inch. As to the cause of the condition. In the first eight cases athetosis was found, being most marked in parts hypertrophic, it was therefore logically inferred that the condition was due to the violence of the spontaneous movements.

I have, however, reported four cases of paradoxical hypertrophy in which no athetosis has ever existed, but in which a severe partial epilepsy was present instead.[2] If the palsy lesion is slight or transient, the nutrition and integrity of the cortical elements remain more or less intact[3], and a post-hemiplegic disorder of motility, such as athetosis or a focal epilepsy, produces in time a hypertrophic condition. The essential factor is the extent and character of the cortical injury, otherwise we would find hypertrophy with every case of choreic paresis or partial epilepsy following palsy lesions. Indeed, extreme atrophy most frequently occurs after choreic paresis or focal epilepsy of hemiplegics. Therefore the association of the peculiar cortical lesion with some degree of post-hemiplegic disorder of motility in parts once paralyzed causes in time the paradoxical pseudo-hypertrophy.

---

[1] A detailed histo-pathological report of the excised hypertrophic tissues is in preparation.

REPORT OF THREE CASES OF PARADOXICAL PSEUDO-HYPERTROPHY IN INFANTILE CEREBRAL HEMIPLEGIA.

Case III. J. P., female; aged twenty-four years, maternal grandfather had chronic rheumatism and was alcoholic. Paternal aunt had idiopathic epilepsy which developed at the menopause. Patient is second in line of birth. The mother had one miscarriage and died of paralysis at fifty-eight years. Pregnancy and labor were normal. There was no paralysis or injury of child at the time of birth. As soon as the patient was able to get about she developed a depraved taste for plaster, clay, muddy water, etc. At 18 months old, during difficult dentition she had a series of convulsions which lasted 12 hours, immediately after which complete left hemiplegia was noted. Nine months from the onset of the hemiplegia the fingers of the left hand developed "cramping spasms" without loss of consciousness, at the time patient commenced to use the left side learning over again how to creep and walk. One year after the hemiplegia she had "falling attacks," attended by a sudden twitching in all the muscles of the left side, during which consciousness was frequently preserved. Patient is feeble-minded; has never attended school but can read and write. The epilepsy probably dates from the hemiplegia, although typical grand mal did not develop until she was 13 years old, during a period of severe menstruation. A sensory aura of numbness on the left side precedes attack. The seizures consist of tonic and clonic convulsions which begin and end on the left (paralyzed) side, as in ordinary spasmodic hemiplegia. Aside from the order of beginning and ending and the abbreviation of the post-paroxysmal stertor, the seizures are typical grand mal in character. The patient also has frequent petit mal, which usually occurs in the morning before rising. The attacks consist of "writhing movements" of the extremities, in which the body and limbs are "thrown in every direction." Grand mal attacks are infrequent (2 or 3 a month), while petit mal attacks occur every three or four days. She has had 25 grand mal attacks in 24 hours. Knee-jerks are exaggerated on the left side and there is considerable loss of power in the left arm and leg. The tongue is deviated to the right on protrusion. Weight 100 lbs., height 5 ft. 2 3-8 in., circumference of left arm (paralyzed) is 1-2 in. larger at biceps than that of the right, while the left forearm is 1-4 in. larger than the right. There is no athetosis or disorder of motility other than the epilepsy in hypertrophic parts and the left face and leg are atrophic. The condition of the hypertrophy is well shown in Fig. III. The measurements are as follows:

---

[2] The epilepsy may also be superimposed upon an athetosis, as in case E. P. of our series.

[3] It is well known also that partial epilepsy and athetosis most frequently occur after slight and transient lesions of the cortex.

	Right	Left
Dynamometric test	44	22
Length from acromion process to external condyle	11 in.	11 in.
Length of internal condyle of humerus to the styloid process of ulna	9 1-2 in.	9 1-2 in.
External condyle to styloid process of radius	9 1-4 in.	9 1-4 in.
Circumference of arm, arm flexed on forearm at right angles	9 in.	9 1-2 in.*
Largest part of forearm in its anatomical position	7 3-4 in.	8in.
Circumference of hand at metacarpophalangeal articulation	7 in.	7 in.
Length of index finger	3 7-16 in.	3 7-16 in.
Length of second finger	4 in.	4 in.
Length of thumb	2 1-2 in.	2 1-2 in.
Circumference of middle finger at middle part of first phalanx	2 in.	2 in.
Circumference of thigh at gluteal fold	20 3-4 in.	20 in.*
Circumference at popliteal	12 in.	11 1-2 in.
Circumference of leg at calf	12 1-2 in.	11 1-4 in.
Dorso-plantar circumference	8 in.	8 in.
Circumference of wrist	5 1-2 in.	5 1-2 in.
Circumference of ankle	7 1-2 in.	7 1-2 in.
Length from ant. sup. spinous process to int. malleolus	31 in.	30 in.

Case IV. A. B., male, aged twenty-three years. Father is intemperate, mother had periodic facial neuralgia and epilepsy; she died from puerperal eclampsia at my patient's birth. Pregnancy and labor were normal in all respects. Patient is the youngest in a family of seven; all the patient's brothers and sisters are well. He always had migraine until his epilepsy began at 14; at this latter age complete left hemiplegia occurred, which was supposed to have been caused by overeating. He had convulsions during the stroke and was prostrated for several months from the physical exhaustion. The left side participates most in the classic grand mal epilepsy which occurs frequently in such cases. The vaso-motor disorders which occur ordinarily in palsy cases are found here also. Although the patient is able to do considerable work with the left hand, there is, nevertheless, a temporarily increased weakness in the left side after convulsions (exhaustion paralysis). Some permanent contractures exist in the flexors of the fingers. No athetotic movements have ever occurred. The power in the paralyzed parts is greater than on the sound side; all reflexes on the left side are exaggerated; dynamometric test shows: left, 48; right, 44. Weight is 136 pounds; height, 5 ft. 3 in. The left arm at the biceps is hypertrophied, the enlargement is 1-4 in. more on the paralyzed side. The remainder of the left side is atrophic.[3]

Case V. A. V., female, aged 36. One maternal aunt was tubercular; another maternal aunt died of epilepsy, and a maternal cousin also died insane. Both parents are very neurotic; the mother still has periodic attacks of neuralgia. The father had epilepsy from "18 months until five years old." A sister of our patient had convulsions at dentition at two, and after a respite of ten years epileptic convulsions began at twelve. She finally died of epilepsy at twenty-seven years of age.

Our patient was born a healthful child at full term. She was, nevertheless, a very "restless and irritable" child. She had scrofula, pertussis and measles. At seven years of age she had scarlet fever, which was followed by nephritis. A few days after the renal complication she had repeated convulsions, high fever and vomiting, which symptoms were followed by a comatose period for an hour. On return to consciousness the entire left side was found paralyzed. Epileptiform attacks began at once. At first the attacks existed as slight "fainting spells" in which the patient fell to the left if standing or sitting.

---

[3] Owing to the patient having passed from our observation after the first examination, we have not been able to furnish a more complete history or a pictorial reproduction of the case.

After a period of five years of hemispastic seizures, classic grand mal developed with the order of invasion and ending in the muscles of the paralyzed side. A sensation of heat rising from the feet to the head constitutes the aura. She has had ten seizures in twenty-four hours; she now averages one attack a month. No athetosis has ever existed in this case. The muscles of the left arm are hypertrophic, the biceps is most hypertrophic, as is well shown on page 647. As the biceps forms almost the entire hypertrophy of the left arm, the relative enlargement in this case, although but 1-4 in., appears much greater than in J. P., in which it was 1-2 in. All the tendon reflexes are much exaggerated on the left side. Weight, 120 1-2 pounds; height 4 ft. 1 3-4 in.; asymmetry of face, palate and cranium, which are development errors due to the hemiplegia rather than stigmata of degeneration, as ordinarily understood. The measurements of the case are as follows:

	Right	Left
Dynamometric test....................	52	19.5
Length from acromion process to external condyle	10 3-4 in.	10 3-4 in.
Length internal condyle of humerus to the styloid process of ulna	9 1-2 in.	9 1-2 in.
External condyle to styloid process of radius	9 1-2 in.	9 1-2 in.
Circum. of arm, arm flexed on forearm at right angle	10 3-4 in.	11 in.*
Largest part of forearm in its anatomical position	8 3-4 in.	8 3-4 in.
Circum. of hand at metacarpo-phalangeal articulations	7 1-4 in.	6 1-2 in.
Length of index finger	3 9-16 in.	3 5-16 in.
Length of second finger	4 in.	3 7-8 in.
Length of thumb	2 1-2 in.	2 5-16 in.
Circum. of middle finger at middle part of first phalanx	2 1-4 in.	2 3-16 in.
Thigh at gluteal fold	22 in.	21 in.
Circum. prosstitial space	14 1-4 in.	14 3-16 in.
Circum. of leg at calf	13 1-2 in.	12 1-4 in.
Dorso-plantar circum.................	8 1-2 in.	8 7-16 in
Circum. of wrist	6 in.	5 7-8 in.
Circum. of ankle	7 7-8 in.	7 3-4 in.
Length of ant. sup. spinous process to int. malleolus......................	31 in.	31 in.

The following is an abstract of the two cases I have previously reported[4].

Case I. E. P., female, age twenty-three years; neurotic history. Although she was born a healthy child at term, labor was difficult and was finished by instruments. She walked at two and had no serious illness until six, when she had severe and prolonged convulsions for 24 to 36 hours. This left the right side completely hemiplegic. Epileptiform crises developed at once and have occurred every two or three days since. They are grand mal, involving the right side first and most. She is feeble-minded. Marked athetosis exists, being most marked in the right arm. The presence of athetosis dates from the day of the stroke. The deep reflexes on the right side are all exaggerated. The following measurements and Fig. I. show well the hypertrophy in this case:

	Right	Left
Weight, 138 lbs., Height, 5 ft. 3 3-4 in.		
Dynamometric test	33	60
Length from acromion process to external condyle	12 in.	12 1-2 in.
Length internal condyle of humerus to the styloid process of ulna	9 1-2 in.	9 1-2 in.
External condyle to styloid process of radius	10 in.	9 1-2 in.
Circumference of arm; arm flexed on forearm at right angles	10 1-4 in	9 1-2 in.
Largest part of forearm in its anatomical position	9 1-4 in.	8 3-4 in.
Circumference of hand at metacarpophalangeal articulation	7 7-16 in.	7 1-2 in.
Length of index finger	3 5-8 in.	3 1-2 in.
Length of second finger	4 1-2 in.	4 in.
Length of thumb	2 5-8 in.	2 1-4 in.
Circumference of middle finger at middle part of first phalanx	2 1-4 in.	2 5-8 in.
Circumference of thigh at gluteal fold	20 1-2 in.	20 1-4 in.
Circumference popliteal space	14 1-4 in.	14 in.
Circumference of leg at calf	13 3-8 in.	13 1-2 in.
Dorso-plantar circumference	8 1-2 in.	8 1-2 in.
Circumference of wrist	6 in.	5 7-8 in.
Circumference of ankle	7 7-8 in.	7 7-8 in.
Length from ant. sup. spinous process to int. malleolus of ankle	32 3-4 in.	33 in.

---

[4] Archives of Neurology and Psychopathology, Vol. 2, Nos. 2 and 3, 1899.

Case II. F. W., female, aged twenty-five years; neurotic family history. Although patient was born a healthy child at term, labor was difficult and instrumental, it being a breach presentation. At two years of age during difficult dentition she had a series of convulsions which left the right side hemiplegic. Epileptiform crises followed the palsy lesion at once. They were convulsive and confined to the right side at first; later they became general, with a right side order of invasion. Patient is feeble-minded, but able to do considerable housework. The whole right side, including the face, is hypertrophic; the bones also enter into the enlargement as shown by skiagraphs in the original publication of the case. Mild scleroderma and Raynaud's disease are also associated in this case. The following measurements and Fig. II. show the hypertrophic state of the hemiplegic parts:

	Right	Left.
Weight, 108 lbs., height 4 ft. 11 3-4 in.		
Dynamometer	46	50
Dynamometer (average) of twelve tests	42 1-2 in.	48 2-3 in.
Length from the acromion process to tip of middle finger	26 1-2 in.	26 1-2 in.
Circumference of biceps (forearm at right angles to arm)...........	10 1-2 in.	9 3-4 in.
Circumference below bent elbow (forearm at L)	10 in.	9 1-4 in.
Circumference at wrist	6 1-2 in.	5 3-4 in.
Circumference behind knuckles	7 1-2 in.	7 1-4 in.
Length of middle finger	4 in.	4 in.
Length of humerus (acromion to ext. condyle; arm bent at L)	11 in.	11 1-2 in.
Olecranon to styloid process (ulnar side)	9 1-4 in.	9 1-4 in.
Circumference at gluteal fold	19 1-4 in.	19 in.
Anterior superior spine to internal malleolus.......................	31 in.	30 1-2 in.

IN RECUMBENT POSITION.

Circumference above knee	13 1-2 in.	13 1-2 in.
Circumference of calf	13 1-2 in.	13 in.
Circumference above ankle	7 1-2 in.	7 1-2 in.
Circumference of arch of foot	8 in.	7 7-8 in.

REFERENCES TO THE CASES.

Gowers: "On Athetosis," etc. Medico-Chirurgical Transactions, 1876. (One Case.)

Bernhardt: "Ueber die von Hammond Athetose gennanten Symptomen Complex." Virchow's Archiv., Bd. 67 (1876).

Bernhardt: "Ein neuer Beitrag zur Lehre von der Athetose." Deutsche med. Woch., Dec. 2, 1876. (Two cases.)

Oulmont: "De l'athétose." Thèse de Paris, 1878. (One case.)

Gaudard: "Contribution a l'etude de l'hémiplégie cérébrale infantile." Geneva, 1884. (One case.) (Not accessible.)

Scheiber: "Ueber einen Fall von Athetosis Spastica." Arch. f. Psychiatrie, xxii (1891). (One case.) (Not accessible.)

Kaiser: "Myotonische Störungen bei Athetose." Neurol. Centralblatt, 1897, p. 674. (One case.)

Clark: "Hypertrophic Infantile Cerebral Palsy and Phocomelus Associated with Epilepsy." Arch. of Neurology and Psychopathology, 1899, Vol. 2, Nos. 3, 4.

IMPORTANT REFERENCES WITHOUT CASES.

Shaw, Clay: "On Athetosis or Imbecility with Ataxia." St. Barthol. Hosp. Reports, 1873.

Dreschfield: "De l'athetosé." Rev. mens. de méd. et de chir., 1878, p. 66.

Berger: "Athétose." Eulenberg's Real-Encyclycopadie, 1885.

Freud und Rie: "Klinische Studie über die halbseitige Cerebrallähmung der Kinder." Wien., 1891.

Brissaud: Encéphalopathies infantiles. Charcot, Traité de méd., Paris, 1894.

Sachs: "Nervous Diseases of Children," 1895, p. 535.

Freud: "Die infantile Cerebrallähmungen," Nothnagel's "Handbuch der specielle Pathologie und Therapie," 1897.